Laminated Root Rot of Douglas-fir

T. W. Childs,[1] and E. E. Nelson[2]

Laminated root rot, caused by the native fungus *Poria weirii,* is widespread in northwestern United States and adjacent Canada: It is especially common and damaging to Douglas-fir. Most native conifers are susceptible but possibly none more so than Douglas-fir.

In Washington and Oregon, this is by far the most destructive root rot of Douglas-fir, causing an estimated annual loss of 32 million cubic feet in the highly productive Douglas-fir subregion alone. Trees of all sizes and ages are attacked, but damage is usually most obvious in stands from about 40 to 125 years old.

The role of *P. weirii* as a "disease of the site" surpasses its importance as a killer of individual trees. Persistence and gradual spread of fungus from one rotation to the next raises the strong possibility of continuing reduction of site productivity.

Description

The disease occurs in patches or centers of infection (fig. 1) often several hundred square feet in extent but sometimes an acre or more. A typical center contains trees that have been dead for diferent periods of time. These trees have usually fallen, but some may be standing. Roots of the downed trees are often broken transversely near the base of the trunk (fig. 2). A few of the living trees in the center may be leaning or may display thin or ragged crowns, poor color, crops of abundant small cones, or decreased terminal and lateral growth. Bark beetles, when attracted to such weakened trees, hasten their death.

Various other pests and disturbances cause somewhat similar openings and crown symptoms but are seldom difficult to distinguish from laminated root rot. For example, in blowdowns and groups of beetle kills where root rot is not involved, most of the

[1] Retired principal plant pathologist Pacific Northwest Forest and Range Experiment Station, USDA Forest Service.

[2] Principal plant pathologist at the PNW Forestry Sciences Laboratory.

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Figure 1.—A laminated root rot center in an 80-year-old Douglas-fir stand.

killing occurs within 1 or 2 years. In shoestring root rot centers, the distinctive white to cream-colored mycelial fans of *Armillaria mellea* can be found between the bark and the wood at bases of dead or weakened trees; however, diagnosis must be especially careful

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Figure 2.—Douglas-fir "rot thrown" as result of attack by *Poria weirii*. Note stubs of decayed roots.

where the more conspicuous *A. mellea* is attacking trees already weakened by *P. weirii*. Root rot caused by *Fomes annosus* may be confused with *P. weirii* in early stages of decay, but typical decay can generally be distinguished by comparison.

Laminated root rot is more reliably identified in the field by the characteristic appearance of the decay. Incipient decay is reddish-brown to brown. In butts and main roots it appears as streaks or broad bands on longitudinal sections and as circular, crescent-shaped, or irregular areas on cross sections (fig. 3). Typical advanced decay is exposed on broken roots of downed trees or can be found by chopping into the base of standing dead trees. This decay is laminated—that is, the wood tends to separate along the annual rings—and it contains numerous oval pockets about one-fiftieth of an inch in diameter and one twenty-fifth of an inch long (fig. 4). Thin, velvety layers or sparse tufts of brown fungal threads are usually present in crevices in the decayed wood; and

thin, brown crusts sometimes form on bark or on surfaces of breaks. In the final stages of decay, the wood becomes a loose, stringy mass that eventually disintegrates completely to leave hollow butts in occasional trees that have survived infection for many years. When *Poria weirii* decay is found, the fungus can be assumed as the primary cause of the opening in the stand.

Fruiting bodies of the fungus are brown crusts dotted with thousands of minute pores. The crusts form on lower sides and in root crotches of downed trees during late summer and early fall. Although fairly common in some years, they are too inconspicuous to be useful in detecting the disease.

Spread

New centers of the disease start, presumably, when spores infect wounds on or near the butts of living trees. Infection within centers spreads from tree to tree

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Figure 3.—Cross section from base of Douglas-fir, showing incipient and typical decay caused by *Poria weirii*.

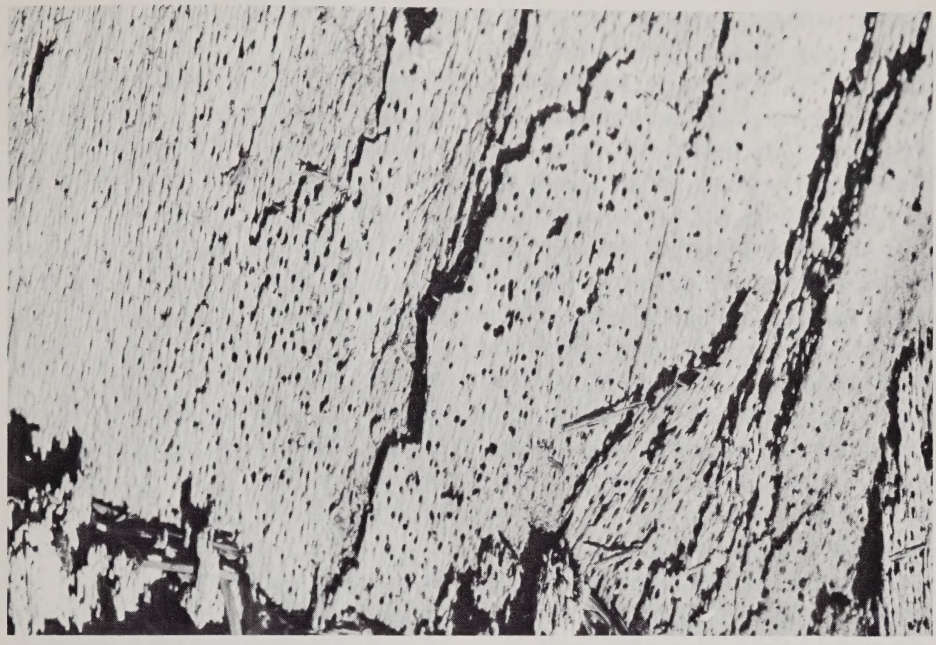

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Figure 4.—Closeup of typical decay caused by *Poria weirii*. Note small pockets and separation of annual rings.

by growth of the fungus along roots. The fungus readily penetrates bark of roots of living trees or trees cut as much as 12 months earlier and colonizes the underlying wood. In forests west of the Cascade Range, fungus growth along roots is vigorous throughout the year. At present, the roles of particular soil, climate, or other site characteristics in development of the disease are unknown, but other soil organisms are probably important in limiting its destructiveness.

In stands of poles or sawtimber, trees within 15 feet of a tree killed by laminated root rot are usually infected. The farther a living tree is from a killed one, the less likely it is to be infected; at distances of 50 feet, infection from the same source is uncommon.

When an infection center is logged or burned, *Poria weirii* continues to live in dead roots as long as 50 years or more. The fungus does not grow through the soil, so if no other food supply grows to it, it eventually dies. If, however, the area is reoccupied by susceptible trees before the fungus has died, some of the roots of the new stand may grow into contact with buried, infectious material. The disease then spreads from root to root, and the infection center reappears in the new stand.

Damage

Poria weirii is important principally as a killer that persists and gradually spreads in a site through successive rotations. The heart rot that it causes in older

trees seldom extends far above the stump and affects yield relatively little. Damage consists almost entirely of loss of actual or potential volumes as younger parts of the stand are destroyed in infection centers.

In young forests, damage increases in geometric proportion to stand age as infection centers gradually become larger and more numerous. Infection is much less abundant in some localities than in others. Predicting where damage will become severe enough to warrant special measures to diminish losses is difficult at present levels of knowledge. As a rough rule of thumb, it may be assumed that damage will be doubled about every 15 years after the first manifestations of the disease. Therefore, when infection centers are common in small pole stands, serious damage must be expected before the trees reach sawtimber size, even though losses to date may have been negligible.

New stands of susceptible species established on previously infected sites can be expected to suffer continuing and increasing mortality. The more prompt and better stocked the regeneration, the more severe this continuing mortality is likely to be. Sites with heavy infection at time of harvest are not likely to produce a well-stocked merchantable stand in succeeding rotations so long as proven methods for controlling the fungus are lacking.

Detection of *Poria weirii* infections in established or planned recreational sites is important. Untrained observers have been known to judge "natural" openings in the forest as suitable for campground or other recreational use when the openings were actually *Poria* infection centers. The hazard of falling trees, of course, renders such areas most unsafe for this purpose.

Control

No practical method of direct control is known or foreseeable, but losses can often be reduced by appropriate management practices. As far as is known, neither clear cutting, partial cutting, nor broadcast burning affect survival of the disease-causing fungus.

When stands are to be clear cut over a period of several years, logging units where root-rot mortality is greatly reducing net increment should be cut first. Units where the highest net increment is still being obtained should be left until last.

In stands not ready for the final cut but suitable for logging of small volumes, all trees on and near larger infection centers or groups of centers may be cut. Values that will otherwise be lost within the next few years can thereby be salvaged. No highly susceptible species such as Douglas-fir should be left, since infected trees seldom respond well to release or live long enough to effectively utilize the added growing space. For the same reason, precommercial thinnings not favoring resistant species offer poor chance for satisfactory return on

investment in heavily infected stands.

Because damage is likely to become increasingly severe when highly susceptible species are used to regenerate areas of previously heavy infection, more resistant species such as hardwoods or western redcedar merit consideration. The chances for these developing into well-stocked, merchantable stands are markedly better than for highly susceptible species such as Douglas-fir. Moreover, a rotation of resistant species (especially hardwoods) will reduce carry-over pockets of *Poria weirii*-infected wood in the soil. The fungus will be deprived of any new, optimum food base or opportunity to spread along highly susceptible roots. Effects of resistant hardwoods on chemical and microbial properties of soils may also reduce survival of residual *Poria weirii*. Before any discussion on species composition is made, both economic and ecological considerations must be carefully weighed.

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